EXHIBIT

"B"

CORNELL UNIVERSITY

NEW YORK PRESBYTERIAN HOSPITAL

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January 28, 2008

Mr. James Butler
591 Summit Avenue
Jersey City, New Jersey 07306

Re: Sabina Paradi

Dear Mr. Butler:

Briefly, the history of the present illness is that Ms. Paradi was a 23-year-old woman who was struck by a pickup truck on 2/25/07. She was treated initially at Saint Vincent's Medical Center. Her initial Glasgow Coma Score was 4, that is, no speech, no eye opening, and reflex (decorticate posturing) movements only. The pupils were unreactive to light. CT scan showed extensive subarachnoid hemorrhage and a right hemisphere subdural hematoma with an 8 mm. right-to-left shift. There were bilateral occipital bone fractures. She had an emergency hemicraniectomy performed on 2/26/07 but postop remained unresponsive with pinpoint pupils that were sluggishly reactive.

The course at Saint Vincent's Medical Center was complicated by pancreatitis thought to be due to Propofol and pneumonia. Her pancreatitis resolved spontaneously. She had a percutaneous enterogastrostomy performed on 3/2/07 and a tracheostomy on 3/7/07. She was transferred to New York-Presbyterian Hospital Columbia Medical Center for continuing care and notes there indicate that she remained unresponsive but with eye opening to stimulation without tracking or response to voice or commands. There was withdrawal to painful stimuli with the limbs bilaterally but no pain localization. During her hospitalization she had many episodes of dysautonomia that were ascribed to her traumatic brain injury and the presence of diffuse axonal injury and preadmission hypoxia. The episodes consisted of increased temperature, heart rate, and blood pressure in the setting of increased tone and posturing of her limbs. She remained at New York-Presbyterian Hospital Columbia Medical Center from 4/18/07 until 5/1/07 when she was transferred to Helen Hayes Hospital for rehabilitation.

She had to be transferred back to New York-Presbyterian Hospital on 5/7/07 because of swelling around her hemicraniectomy in addition to recurrent episodes of dysautonomia. While she was at New York-Presbyterian Hospital, her parents agreed to have her made comfort care only because of her poor prognosis for recovery of consciousness and she died on 6/30/07.

The following records were reviewed:

1. Autopsy Report. An autopsy was performed at The Office of the Chief Medical Examiner of The City of New York on 7/10/07. On general examination it was noted that she had a palpable subcutaneous ventriculoperitoneal shunt inserted into the left frontal parietal scalp region that was also visible over the left side of the neck, the left side of the chest, and the upper right side of the abdomen. There was a circular skull defect (1.4") over the left frontal parietal skull and the VP shunt entered the brain through the dura. There was a 7" C-shaped surgical scar over the right parietal region with an underlying prosthetic cranioplasty. Cranioplasty had clips in place in the native skull without tissue overgrowth. There was an irregular pink scar (1/4") over the occipital region just to the right of the midline. There were healing nondisplaced hairline fractures across the left posterior fossa and the right posterior fossa. A tracheostomy tube was placed and there was a percutaneous endoscopic gastrostomy tube over the left side of the abdomen that was in place in the stomach.

The brain weighed 1450 grams, showed no evidence of epidural or recent subdural hemorrhage. There was no evidence of a cervical spine fracture. The heart was normal but the lungs, in their posterior aspect, were dark purple, firm, and edematous.

On microscopic examination the following findings were notable: In the heart there was focal contraction band necrosis, fatty infiltration of the right ventricle. In the lungs was evidence of pneumonia with focal intraalveolar macrophages, slight focal intraalveolar inflammatory cells. They were mainly mononuclear and there were focal peribronchial and interstitial inflammatory cells, also mainly mononuclear cells.

Cerebrospinal fluid was cultured and was positive for staphylococcus that was coagulace negative. Blood and cerebrospinal fluid grown in a blood culture bottle were negative for any organisms.

Toxicology performed on the vitreous humor of the eye was positive for Fentanyl and morphine which had been given therapeutically antemortem.

Neuropathological examination of the brain revealed golden yellow discoloration of the orbital surface of the left frontal lobe with softening of various gyri, namely the gyri recti and the medial orbital gyri. The right occipito-temporal and right temporal pole gyri were also softened. There was disruption and nonhemorrhagic necrosis (death of tissue) in the gyri mentioned above. There were no focal lesions of the nuclear structures of the brain nor was there any shift of the midline structures.

The spinal cord dura showed evidence of a subdural hemorrhage in the thoracic region measuring 7x1.2 cm. Transverse sections showed nonhemorrhagic softening of the midthoracic spinal cord measuring 5 cm. in length. (**Comment:** These findings indicate that there was a spinal cord injury that would have led to paraplegia had the patient survived.)

Microscopic sections showed necrosis, gliosis (that is, scarring), and macrophages (infiltration of inflammatory cells) of the right thalamus and the periventricular gray matter nuclei in the medulla. The hippocampus showed neuronal loss and gliosis. Microscopic sections through the gyri previously mentioned showed necrosis with many microphages, vascular proliferation, gliosis and foci of hemosiderin pigment (remnant of the breakdown of red cells).

The diagnosis was traumatic brain injury, remote; status post craniectomy with skull prosthesis and ventriculostomy; subdural hemorrhage organized over the cerebral convexities and spinal cord; subarachnoid hemorrhage organized over the left frontal lobe; cortical and white matter contusions, frontal and temporal lobes; chronic diffuse anoxic-ischemic encephalopathy with neuronal loss and gliosis; organizing microinfarcts and focal cortical necrosis of the left frontal lobe.

There is "supplemental case information" appended to this report. The source of the information was a member of the resident staff of the CCU at New York-

Presbyterian Hospital Columbia Medical Center, a doctor E. Gilmore. The report summarizes her stay and her course and is remarkable for the following: "Despite efforts and being transferred from hospital to rehab and back, she remained in a vegetative state throughout her hospital course. She was given a 1% chance of recovering. Her family decided on extubating her and she was given comfort care."

2. Records of Saint Vincent's Catholic Medical Centers of New York – Manhattan. Of note, these records are the results of a CT scan of the head performed without contrast on 2/25/07 and following craniectomy on 3/14/07. There 3/14/07 CT scan documents infarcts within the right posterior temporal lobe and bilateral occipital lobes, larger on the left. There was prolapse of the brain through the right fronto-parietal craniotomy. There was persistence but decrease in density of the hemorrhage involving both frontal lobes and there was a decrease in the previously-noted left-to-right shift of the brain under the falx. Previous CT scans had been performed on 2/25, 2/26 immediately following the right fronto-parietal craniectomy and evacuation of the right convexity subdural hemorrhage, and also on 2/28/07. Of note is the fact that these CT scans also showed evidence of multiple skull fractures including a left temporal bone fracture, bilateral occipital bone fractures, bilateral sphenoid bone clivus, and left petrous portion of the temporal bone fractures. The report of the 2/26 CT scan raises the possibility that the petrous bone fractures may impinge on the internal carotid arteries bilaterally.

A CT scan of the cervical spine without contrast performed 2/25/07 showed no evidence of cervical spine fracture of subluxation. CT scan with contrast performed on 3/2/07 did not show any evidence of vascular abnormalities. CT scan on 3/6/07 showed placement of VP shunt for hydrocephalus following subarachnoid hemorrhage. Twenty-one channel EEG performed 4/3/07 showed low amplitude and diffusely-slow polymorphic delta activity. (**Comment:** CT scans of the brain taken immediately after the head trauma and subsequently while the patient was at Saint Vincent's Catholic Medical Center in Manhattan document the severity of the head trauma and the diffuse brain injury. The electroencephalogram performed on 4/3/07 prior to transfer to New York-Presbyterian Hospital confirms the diffuse nature of the injury and is not compatible with a conscious state.

3. Admission, history and physical from Helen Hayes Hospital. Admission date was 5/1/07. The admission history reviews the present illness and lists the

medications the patient was on at the time of transfer from New York-Presbyterian Hospital Columbia Medical Center. On neurological examination, the admitting physician, Dr. Glenn Seliger, noted on mental status "she does not alert to voice or follow commands. She may show some nonspecific arousal to voice. She does not track or clearly blink to threat." On motor exam he found "little or no significant movement even to pain. The hands tend to be kept in more of an extensor posture. Legs are currently braced and more difficult to assess but only trace movement in the toes is noted."

A discharge summary dated 5/7/07 indicates that while at Helen Hayes Hospital the patient's PEG became clogged and was discontinued. Her Depakote, an antiseizure drug, had to be discontinued because of abnormal liver function tests. Swelling around the right side of the head necessitated transfer back to New York-Presbyterian Hospital. (**Comment:** These records indicate that as of 5/1/07 Ms. Paradi had not recovered consciousness and remained in a vegetative state.)

Records from New York-Presbyterian Hospital Columbia Medical Center from 4/18-07 – 5/1/07 and from 5/7/07 – 6/30/07. Following transfer to Columbia Presbyterian, she had repeated CT scans of her head and an MRI of the brain that showed the presence of extensive injury. On an MRI on 4/18/07, there was bulging of the right frontal and parietal lobes through the boney defect of the previous hemicraniectomy. There were bilateral frontal lobe parenchymal hematomas still evident. There was also parenchymal bleeding still present in the left posterior lateral frontal lobe, the posterior lateral right temporal lobe, and the anterior right temporal lobe. There was a subdural collection along the right side of the tentorium and a small rightsided extra-axial collection. There was "extensive FLAIR hyperintensity involving the frontal regions bilaterally...adjacent to the posterior left frontal hematoma likely related to a combination of encephalomalacia (brain softening), edema (brain swelling), and/or gliosis (scarring of the brain tissue)....Gradient echo images revealed small petechial hemorrhages involving the subcortical white matter primarily in the temporal lobes bilaterally as well as the posterior midbrain and the pons and the splenium of the left corpus collosum suggestive of diffuse axonal injury.... In addition, there was evidence of hydrocephalus, intraventricular hemorrhage in the right occipital horn and enlargement of the left lateral ventricle related to exvacuo dilation caused by loss of left frontal lobe brain tissue. A repeat EEG on 4/22/07 showed "severe, diffuse, background slowing and attenuation more prominent on the right indicative of

diffuse cerebral dysfunction worse on the right...." Continuous monitoring on over several days, 4/18-22/07 showed similar findings. (**Comment:** The MRI scans confirm the presence of diffuse brain injury involving both the cortex and the brainstem. The EEG showed slowing and low voltage nature of the electrical output of the brain confirming the absence of consciousness.).

4, Multiple notes from the Neuro ICU attendings and residents confirm that the patient had no evidence of consciousness. On 4/21/07 the Neuro ICU attending wrote that the patient breathed spontaneously, that her pupils were reactive, and that her eyes were open but that she had no response to command. She may have blinked to threat on the right but not on the left. She had opistotonic posturing. (Opistotonic posturing is extensor or decerebrate posturing again is not compatible with conscious state.)

On 4/23 it was noted that she had spontaneous, saccadic EOMs but was rigid and spastic in all four extremities. On 4/24/07 a note states that the patient is vegetative with eyes open. On 4/29/07 spontaneous eye opening was noted but not to stimulation.

Subsequently, at the end of June, 2007, because of the patient's failure to regain consciousness and her poor prognosis, the family allowed her to be comfort care only and she was allowed to die without intervention.

5. A report by Steven R. Flannigan, a physiatrist at Mount Sinai Hospital in New York dated November 26, 2007 and addressed to Michael Kaplen. Dr. Flannigan reviewed medical records of Saint Vincent's, New York-Presbyterian Hospital, Helen Hayes Hospital, as well as a brief report from Angelo R. Canado, Ph.D. Dr. Flannigan noted that "on several occasions she was noted to be agitated...." He cites an occupational therapist note of 3/27, a nurse's note of April 6. (**Comment:** These episodes were autonomic "storms" that were the seizure-like discharges of a damaged brain, not related to any conscious agitation or upset. He also mentions that there were Neurology and Neurosurgical notes that noted that the patient intermittently followed simple commands and was noted to move her left upper limb purposefully. I have quoted notes immediately after these from the Neuro ICU attendings which indicate that the patient had only decerebrate, reflex posturing and that she was rigid

and spastic with contractures of all limbs and, therefore, could not have moved purposefully as described.

On the basis of these findings, Dr. Flannigan concludes that the patient was in a minimally conscious state. The minimally conscious state he correctly differentiates from the vegetative state by stating such a patient has an intermittent ability to demonstrate purposeful activity in response to internal or external stimulation. He bases this on her intermittent agitation which has been explained, he infers the presence of the minimally conscious state from the presence of agitation which is a misinterpretation of her disautonomia and movements of her left upper limb which were interpreted as purposeful on three occasions and following simple commands on two occasions. These observations were not confirmed by the patient's ICU attendings and it was the opinion of the doctors taking care of her that she never manifested any evidence of consciousness.

6. A report of Angnelo R. Canado, a licensed psychologist, who visited the patient on 6/19/07. He saw the patient to screen her for potential consideration for coma stim/coma recovery program following discharge. He did not review the chart. He did not discuss her medical condition with any of the doctors caring for the patient. He obtained information from the patient's parents. He spent fifteen minutes in the ICU and said that he could not comprehensively evaluate the patient. He reviewed the history and he mentioned she presented as unresponsive and demonstrated decorticate posturing upon contact. It was his impression that the patient was a potential but marginal candidate for the coma stimulation program. His impression was that the patient was in a vegetative state and might be able to progress to the minimally responsive state over a period of time.

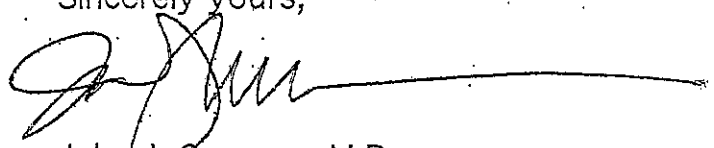
Comment: My medical impression upon review of these records is that Sabina Paradi suffered a severe and diffuse traumatic brain injury on 2/25/07 that rendered her unconscious from the moment of impact until her death on June 30, 2007. At some point subsequent to the head injury, she evolved from coma (a sleep-like state from which the patient cannot be awakened) to the vegetative state (a state of wakefulness without consciousness). Imaging studies performed periodically during her course showed evidence of severe structural damage to the brain: EEGs, both at Saint Vincent's and at New York-Presbyterian Hospital showed low voltage and diffuse slowing indicative of severe brain damage without consciousness.

Her autopsy, performed at the Medical Examiner's Office on 7/10/07, confirmed the presence of "chronic, diffuse, anoxic ischemic encephalopathy with neuronal loss and gliosis as well as focal cortical necrosis of the left frontal lobe with cortical and white matter contusions of both frontal and temporal lobes as well as necrosis and scarring of the basal ganglia (right thalamus) and the brainstem (medullary periventricular gray matter nuclei)."

I, therefore, must conclude that the allegations that the patient on some days in April followed commands or moved her left side purposefully or was agitated represent misinterpretations of reflex postures. It is not possible for a patient who suffered the injuries that Ms. Paradi suffered to have any conscious experience. It was in recognition of this fact that her physicians at New York-Presbyterian Hospital recommended that she be made DNR and be withdrawn from aggressive care.

I find with a reasonable degree of medical certainty that Sabina Paradi sustained no conscious pain and suffering from the time of the trauma on 2/25/07 until her death on 6/30/07.

Sincerely yours,



JJC:CH

John J. Caronna, M.D.